Amendments to the Specification

Please replace paragraphs 0009, 0012, 0019, 0020 (Table 2) and 0021 (Table 2) as identified in the published application with the following amended paragraphs:

[0009] While not desiring to be bound to any specific theory, we conclude that one or more of the several types of calcium-permeable CNS ion channels mentioned below can be involved in controlling such migration, including: a) the various aspects of the NMDA (N-methyl-D-aspartate) receptor channel complex; b) the voltage-dependent Ca[[.sup.2+]]$^{2+}$ channels; and c) other channels directly coupled to glutamate (or excitatory amino acid) receptors. Such channels are reviewed in: Sommer, B. and Seeburg, P. H. "Glutamate receptor channels: novel properties and new clones" Trends Pharmacological Sciences 13:291-296 (1992); Nakanishi, S., "Molecular Diversity of glutamate receptors and implications for brain function", Science 248:597-603 (1992).

[0012] Other compounds that are useful in the invention include voltage-dependent calcium channel antagonists, e.g. those which exert a substantial direct effect on glutamate toxicity mediated by the L-type voltage dependent Ca[[.sup.++]]$^{++}$ channel in that they produce a statistically significant result in experiments measuring glutamate induced effects by the general method described in Karschian and Lipton, J. Physiol.418:379-396 (1989) or by other techniques for measuring antagonism of the L-type Ca[[.sup.++]]$^{++}$ channel known to those in the art. (We contrast the direct effect so measured with the secondary effects of excitoxicity mediated by other channels, which in turn causes flow through the voltage dependent Ca[[.sup.++]]$^{++}$ channels.) Particular candidate compounds include Class I voltage dependent Ca[[.sup.++]]$^{++}$ channel antagonists, e.g., phenylalkylamines.

[0019] Table 1, below, lists various suitable NMDA and non-NMDA receptors which do not operate via the voltage-dependent Ca[[.sup.++]]$^{++}$ ion channel. Tables 2-4 list antagonists of the voltage dependent Ca[[.sup.++]]$^{++}$ channel, which can be used by themselves in connection with the first aspect of the invention, and which can also be used in combination with other antagonists in the second aspect of the invention.

[0020]

TABLE 2

Antagonists of the Voltage Dependent Calcium Channels (N, L, T, P and other types)
dihydropyridines
(e.g., nimodipine)
phenylalkylamines
(e.g., verapamil, (S)-emopamil, D-600, D-888)
benzothiazepines
(e.g., diltiazem and others)
bepridil and related drugs
diphenylbutylpiperdines
diphenylpiperazines
(e.g., flunarizine/cinnarizine series)
HOE 166 and related drugs
fluspirilene and related drugs
toxins and natural compounds
(e.g., snail toxins -- ~~.omega.~~ω–conotoxin GVIA and GVIIA, maitotoxin, taicatoxin, tetrandine, hololena toxin, plectreurys toxin, funnel-web spider venom and its toxin fraction, agatoxins including ~~.omega.~~ω-agatoxin IIIA and ~~.omega.~~ω-agatoxin IVA.

[0021]

~~TABLE 2~~

~~Antagonists of the Voltage Dependent Calcium Channels (N, L, T, P and other types)~~
~~dihydropyridines~~
~~(e.g., nimodipine)~~
~~phenylalkylamines~~
~~(e.g., verapamil, (S)-emopamil, D-600, D-888)~~

~~benzothiazepines~~

~~(e.g., diltiazem and others)~~

~~bepridil and related drugs~~

~~diphenylbutylpiperdines~~

~~diphenylpiperazines~~

~~(e.g., flunarizine/cinnarizine series)~~

~~HOE 166 and related drugs~~

~~fluspirilene and related drugs~~

~~toxins and natural compounds~~

~~(e.g., snail toxins --.omega.conotoxin GVIA and GVIIA, maitotoxin, taicatoxin, tetrandine, hololena toxin, plectreurys toxin, funnel-web spider venom and its toxin fraction, agatoxins including .omega.-agatoxin IIIA and .omega.-agatoxin IVA.~~

TABLE 3

Dihydropropyridine Calcium Chanel Antagonists	
nifedipine	KW3049
niludipine	oxodipine
PY108-068 (darodipine	CD349
mesudipine	TC81
GX1048	YM-09730-5 or (4S)DHP
floridine	MDL72567
nitrendipine	Ro18-3981
nisoldipine	DHP-218
nimodipine	nilvadipine
nicardipine	amlodipine
felodipine	8363-S
PN200-110 (Isradipine)	iodipine
CV4093	azidopine